2:30

718-3 Effects of Smoking Status on Long-Term Mortality, Morbidity and Need for Additional Coronary Interventions After Successful Percutaneous Coronary Revascularization

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The aim of this study was to examine the effect of smoking status on outcome after percutaneous coronary revascularization (PCR). All patients after successful PCR were followed over 16 years (mean 4.5 ± 3.7 years). Patients were divided into 4 groups: nonsmokers (I, n = 2009), former smokers (II, n = 2259), quitting smokers immediately after PCR (III, n = 435) and persisting smokers (IV, n = 734). At baseline, smokers (III and IV) were younger than I and II, had shorter duration of angina and fewer comorbid conditions, but had more prior myocardial infarction. Smokers had less extensive coronary artery disease and prior coronary bypass surgery (CABG) and higher rates of complete revascularization.

After adjustment for baseline characteristics, smokers had greater risk of death (RR = 1.20 and RR = 1.74, $p < 0.001$) and Q-wave infarction (RR = 1.44 and RR = 2.08, $p = 0.11$), but less of PCR (RR = 0.80 and RR = 0.67, $p < 0.001$) and CABG (RR = 0.71 and RR = 0.68, $p = 0.002$) relative to non-smokers. By 7 years, patients who continued to smoke after PCR had greater risk of death than those who quit immediately (RR = 1.48, $p = 0.04$).

Conclusion: Accounting for their favorable clinical and angiographic profile, smokers are at greater risk of death or Q-wave infarction after successful PCR. Smokers have fewer revascularization procedures during follow-up. This study demonstrates that quitting smoking after PCR reduces risk of death by 33%.

2:45

718-4 Lessons from the West of Scotland Coronary Prevention Study (WOSCOPS): Who should be treated

J. Shepherd for the WOSCOPS Publications Committee. *University Department of Pathological Biochemistry, Glasgow Royal Infirmary, Glasgow G4 OSF, USA*

WOSCOPS recently defined the benefits of pravastatin therapy in the prevention of coronary heart disease (CHD) events in middle-aged hypercholesterolemic men without prior myocardial infarction. Individuals at highest risk exhibited the greatest accumulation of events and gained most from treatment.

The significant univariate predictors of outcome were smoking habit, diabetes mellitus, history of hypertension, nitrate consumption, minor ECG abnormality, angina pectoris, family history of CHD, widowhood, educational achievement and employment status. Continuous variables of predictive significance were age, height, blood pressure, VLDL cholesterol, HDL cholesterol, log [Triglyceride] and total cholesterol/HDL cholesterol ratio.

The above elements were used to calculate individual risk scores and thereby to determine the probability of a major coronary event during the five year follow-up period of the study. On the strength of this we concluded that targeted screening is most cost effective in identifying at-risk patients. Compared with the untargeted approach, the former would identify, in the top quartile of the WOSCOPS risk distribution, 45% of the primary endpoints and 64% of the CHD deaths. These results reinforce the merits of the new joint European and NCEP guidelines for the preventive management of CHD.

3:00

718-5 Lipoprotein(a), Lipids, Aspirin and Risk of Myocardial Infarction in the Physicians' Health Study

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Previously reported data from the Physicians' Health Study (PHS) indicate no association between lipoprotein(a) (Lp[a]) and subsequent risk of myocardial infarction (MI) among a large cohort of middle-aged men followed for an average of 60.2 months. In a follow-up analysis of 296 confirmed cases of MI and 296 controls, we evaluated for evidence of association between Lp(a) and cardiovascular risk among those with and without hyperlipidemia and among those randomized to aspirin and placebo.

Among those with total cholesterol (Tc) > = 200 mg/dL, the age-and smoking-adjusted relative risks (RRs) of first MI associated with Lp(a) level above the 25th, 50th, 75th, 90th, and 95th percentile of the control distribution were 0.9, 1.1, 1.6, 1.7, 1.0; (all p-values nonsignificant, p-trend = 0.5). Among those with total cholesterol <200 mg/dL, the adjusted relative risks of MI associated with these cutpoints were 0.9, 0.7, 1.2, 0.7, 1.4; (all p-values nonsignificant, p-trend = 0.7). Analyses limited to those with cholesterol > = 240 mg/dL or to individuals with elevated Tc/HDL ratios revealed similar null findings.

Among those randomized to aspirin, the age-and smoking-adjusted relative risks (RRs) of first MI associated with Lp(a) levels were 0.9, 0.9, 1.5, 1.5, 1.0; (all p-values nonsignificant, p-trend = 0.9). Among those randomized to placebo, the age-and smoking-adjusted RRs of first MI associated with Lp(a) levels were 0.8, 0.9, 1.4, 1.5, 1.3; (all p-values nonsignificant, p-trend = 0.9). No significant interaction was observed between Lp(a) and aspirin on risk of first MI.

These data indicate that lipid parameters and aspirin use did not modify the lack of overall effect of Lp(a) on the risk of first myocardial infarction in the Physicians' Health Study.

3:15

718-6 Red Wine, White Wine, Liquor, Beer, and Risk of Coronary Hospitalizations

A.L. Klatsky, M.A. Armstrong, G.D. Friedman. *Kaiser Permanente Medical Care Program, Oakland, CA, USA*

International comparison data suggest that wine is more protective against coronary heart disease (CHD) than beer or liquor, and there are potentially protective antioxidants in wine, especially red wine. Yet prospective population studies show no consensus about this issue. We studied alcoholic beverage choice in relation to later CHD hospitalizations among 128,934 persons (n hospitalized = 3931). Cox proportional hazards models with 9 covariates were used, plus a variable for drinks/day of each beverage type. Categories of wine drinkers included red only, white only, both red and white and "other". Total alcohol drinking was inversely related to CHD risk ($p < 0.001$) in each sex. In multivariate models uncontrolled for total alcohol, each beverage type showed apparent CHD protection; relative risks (RR) per drink/day for all persons follow: all wine = 0.8 ($p < 0.01$); liquor = 0.9 ($p < 0.05$); beer = 0.7 ($p < 0.001$). Controlled for total alcohol, these relationships lost significance (RR = 0.9 for wine or beer; RR = 1.1 for liquor; $p > 0.05$). Controlled for total alcohol, the RR's for the wine subsets follow: red only = 1.2 ($p > 0.05$); white only = 1.0 ($p > 0.05$); red and white = 0.8 ($p < 0.05$); other wine = 0.08 ($p > 0.05$). Wald chi-square tests showed no significant CHD risk differences between beverage types and/or wine subsets. These data support the conclusion that alcohol drinking protects against CHD primarily through effects of ethyl alcohol, because risk differences between beverage types are minor.

719 Stents: In-Stent Restenosis I

Monday, March 17, 1997, 4:00 p.m.–5:30 p.m.
Anaheim Hilton and Towers, Pacific C

4:00

719-1 Morphologic and Procedural Predictors of Diffuse In-Stent Restenosis

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To understand the process of diffuse in-stent restenosis, we analyzed the clinical, procedural, quantitative angiographic {QCA, including lesion length and reference and minimum lumen diameters (MLD), in mm}, and serial (post-intervention and follow-up) intravascular ultrasound {including reference, stent, lumen, and neointimal tissue (stent-lumen) areas, in mm^2} in 201 stented lesions. An injury score (IS) was constructed: 0 (PTCA pressure ≤16 atm & balloon:artery ratio ≤1.1), 1 (PTCA pressure >16 atm or balloon:artery ratio >1.1, and 2 (PTCA pressure >16 atm & balloon:artery ratio >1.1). Lesions were classified as *no* (neointimal tissue ≤75% stent area), *focal* (neointimal tissue >75% stent area, over ≤ 10 mm stent length), and *diffuse* restenosis (neointimal tissue >75% stent area, over >10 mm stent length). Univariate predictors included:

	Non (n = 56)	Focal (n = 63)	Diffuse (n = 82)	p ANOVA
QCA				
Lesion length	6.20 ± 5.65	7.89 ± 5.13	10.67 ± 7.00	0.0219
Reference lumen	3.31 ± 0.77	2.87 ± 0.52	2.71 ± 0.55	< 0.0001
Final MLD	3.01 ± 0.52	2.78 ± 0.56	2.56 ± 0.61	0.0002
Ultrasound				
Reference lumen	12.75 ± 6.90	8.44 ± 2.15	8.40 ± 3.2	< 0.0001
Stent area	9.61 ± 3.98	7.08 ± 2.57	6.73 ± 2.19	< 0.0001
IS: 0/1/2 (%)	27/73/0	15/68/19	20/51/29	0.0001

MONDAY ORAL